

029654

L Number	Hits	Search Text	DB	Time stamp
1	9	ARL4	USPAT; US-PGPUB; EPO; DERWENT; IBM_TDB	2004/02/16 13:34
2	11	ARL adj "4"	USPAT; US-PGPUB; EPO; DERWENT; IBM_TDB	2004/02/16 13:34
3	1	ARF adj like adj protein adj "4"	USPAT; US-PGPUB; EPO; DERWENT; IBM_TDB	2004/02/16 13:34
4	0	ARF adj like adj "4"	USPAT; US-PGPUB; EPO; DERWENT; IBM_TDB	2004/02/16 13:35
5	0	ADP adj ribosyl\$10 adj factor adj like adj protein adj (ARF adj like adj "4")	USPAT; US-PGPUB; EPO; DERWENT; IBM_TDB	2004/02/16 13:36
6	0	ADP adj ribosyl\$10 adj factor adj like adj (ARF adj like adj "4")	USPAT; US-PGPUB; EPO; DERWENT; IBM_TDB	2004/02/16 13:39
8	21	ARL4 or (ARL adj "4") or (ARF adj like adj protein adj "4") or (ARF adj like adj "4") or (ADP adj ribosyl\$10 adj factor adj like adj protein adj (ARF adj like adj "4")) or (ADP adj ribosyl\$10 adj factor adj like adj (ARF adj like adj "4"))	USPAT; US-PGPUB; EPO; DERWENT; IBM_TDB	2004/02/16 13:39
9	22289	(435/6).CCLS.	USPAT; US-PGPUB; EPO; DERWENT	2004/02/16 13:40
11	7606	(435/7.1).CCLS.	USPAT; US-PGPUB; EPO; DERWENT	2004/02/16 13:41
12	2230	(435/7.21).CCLS.	USPAT; US-PGPUB; EPO; DERWENT	2004/02/16 13:41
13	875	(435/7.24).CCLS.	USPAT; US-PGPUB; EPO; DERWENT	2004/02/16 13:41
14	615	(435/21).CCLS.	USPAT; US-PGPUB; EPO; DERWENT	2004/02/16 13:41
15	1921	(435/29).CCLS.	USPAT; US-PGPUB; EPO; DERWENT	2004/02/16 13:41
16	1642	(436/63).CCLS.	USPAT; US-PGPUB; EPO; DERWENT	2004/02/16 13:41
17	768	(436/86).CCLS.	USPAT; US-PGPUB; EPO; DERWENT	2004/02/16 13:41

18	2487	(436/501).CCLS.	USPAT; US-PGPUB; EPO; DERWENT	2004/02/16 13:42
19	284	(436/504).CCLS.	USPAT; US-PGPUB; EPO; DERWENT	2004/02/16 13:42
21	33266	((435/6).CCLS.) or ((435/7.1).CCLS.) or ((435/7.21).CCLS.) or ((435/7.24).CCLS.) or ((435/21).CCLS.) or ((435/29).CCLS.) or ((436/63).CCLS.) or ((436/86).CCLS.) or ((436/501).CCLS.) or ((436/504).CCLS.)	USPAT; US-PGPUB; EPO; DERWENT	2004/02/16 13:43
22	2	(ARL4 or (ARL adj "4") or (ARF adj like adj protein adj "4") or (ARF adj like adj "4") or (ADP adj ribosyl\$10 adj factor adj like adj protein adj (ARF adj like adj "4")) or (ADP adj ribosyl\$10 adj factor adj like adj (ARF adj like adj "4"))) and (((435/6).CCLS.) or ((435/7.1).CCLS.) or ((435/7.21).CCLS.) or ((435/7.24).CCLS.) or ((435/21).CCLS.) or ((435/29).CCLS.) or ((436/63).CCLS.) or ((436/86).CCLS.) or ((436/501).CCLS.) or ((436/504).CCLS.))	USPAT; US-PGPUB; EPO; DERWENT	2004/02/16 13:43

029654

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NEWS 7 OCT 21 BIOSIS file reloaded and enhanced
NEWS 8 OCT 28 BIOSIS file segment of TOXCENTER reloaded and enhanced
NEWS 9 NOV 24 MSDS-CCOHS file reloaded
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NEWS 19 DEC 22 ABI-INFORM now available on STN
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NEWS 21 JAN 27 A new search aid, the Company Name Thesaurus, available in
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NEWS 22 FEB 05 German (DE) application and patent publication number format
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FILE 'HOME' ENTERED AT 13:47:03 ON 16 FEB 2004

=> file ca

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

0.21

0.21

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FILE COVERS 1907 - 12 Feb 2004 VOL 140 ISS 8

FILE LAST UPDATED: 12 Feb 2004 (20040212/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> S ARL4

L1 13 ARL4

=> SARL(W)4

SARL(W)4 IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system.

For a list of commands available to you in the current file, enter "HELP COMMANDS" at an arrow prompt (=>).

=> s ARF(W)like(W)protein(W)4

6089 ARF

202 ARFS

6101 ARF

(ARF OR ARFS)

554718 LIKE

168 LIKES

554861 LIKE

(LIKE OR LIKES)

1556730 PROTEIN

1077314 PROTEINS

1800744 PROTEIN

(PROTEIN OR PROTEINS)

4721075 4

L2 1 ARF(W)LIKE(W)PROTEIN(W)4

=> s ARF(W)like(W)4

6089 ARF

202 ARFS

6101 ARF

(ARF OR ARFS)

```

554718 LIKE
168 LIKES
554861 LIKE
      (LIKE OR LIKES)
4721075 4
L3      2 ARL(W) LIKE(W) 4

=> s ARL(W) 4
      649 ARL
      37 ARLS
      668 ARL
      (ARL OR ARLS)
4721075 4
L4      2 ARL(W) 4

=> s ADP(W)ribosyl10(W)factor(W)like(W)protein(W)4
10(W)FACTOR(W)LIKE(W)PROTEIN(W)4 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s ADP(W)ribosylation(W)factor(W)like(W)protein(W)4
      55846 ADP
      80 ADPS
      55893 ADP
      (ADP OR ADPS)
      5602 RIBOSYLATION
      29 RIBOSYLATIONS
      5604 RIBOSYLATION
      (RIBOSYLATION OR RIBOSYLATIONS)
      792260 FACTOR
      698766 FACTORS
      1249791 FACTOR
      (FACTOR OR FACTORS)
      554718 LIKE
      168 LIKES
      554861 LIKE
      (LIKE OR LIKES)
      1556730 PROTEIN
      1077314 PROTEINS
      1800744 PROTEIN
      (PROTEIN OR PROTEINS)
      4721075 4
L5      3 ADP(W)RIBOSYLATION(W)FACTOR(W)LIKE(W)PROTEIN(W)4

=> s ADP(W)ribosylation(W)factor(W)like(W)4
      55846 ADP
      80 ADPS
      55893 ADP
      (ADP OR ADPS)
      5602 RIBOSYLATION
      29 RIBOSYLATIONS
      5604 RIBOSYLATION
      (RIBOSYLATION OR RIBOSYLATIONS)
      792260 FACTOR
      698766 FACTORS
      1249791 FACTOR
      (FACTOR OR FACTORS)
      554718 LIKE
      168 LIKES
      554861 LIKE
      (LIKE OR LIKES)
      4721075 4

```

```

L6          5 ADP(W) RIBOSYLATION(W) FACTOR(W) LIKE(W) 4
=> s 11 or 12 or 13 or 14 or 15 or 16
L7          18 L1 OR L2 OR L3 OR L4 OR L5 OR L6

=> s macrophage or phagocyt or monocyte
=> s macrophage or phagocyt or monocyte
    77254 MACROPHAGE
    59981 MACROPHAGES
    94175 MACROPHAGE
        (MACROPHAGE OR MACROPHAGES)
    1 PHAGOCYT
    32228 MONOCYTE
    25266 MONOCYTES
    40615 MONOCYTE
        (MONOCYTE OR MONOCYTES)
L8          118265 MACROPHAGE OR PHAGOCYT OR MONOCYTE

=> del 18
DELETE L8? (Y)/N:y

=> s macrophage or phagocy? or monocy?
    77254 MACROPHAGE
    59981 MACROPHAGES
    94175 MACROPHAGE
        (MACROPHAGE OR MACROPHAGES)
    32548 PHAGOCY?
    57093 MONOCY?
L8          151569 MACROPHAGE OR PHAGOCY? OR MONOCY?

=> s activat? or inhibi? or modulat?
    1070254 ACTIVAT?
    1602347 INHIBI?
    263698 MODULAT?
L9          2588082 ACTIVAT? OR INHIBI? OR MODULAT?

=> s 18 or 19
L10         2660591 L8 OR L9

=> s 17 and 110
L11         9 L7 AND L10

=> d 111 1-9 bib ab

L11 ANSWER 1 OF 9  CA  COPYRIGHT 2004 ACS on STN
AN  140:54198  CA
TI  Lectin-related resistance factors against bruchids evolved through a
    number of duplication events
AU  Lioi, L.; Sparvoli, F.; Galasso, I.; Lanave, C.; Bollini, R.
CS  Istituto di Genetica Vegetale, CNR, Bari, 70126, Italy
SO  Theoretical and Applied Genetics (2003), 107(5), 814-822
    CODEN: THAGA6; ISSN: 0040-5752
PB  Springer-Verlag
DT  Journal
LA  English
AB  Abundant lectin-related proteins found in common beans (Phaseolus vulgaris
    L.) have been shown to confer resistance against the larvae of a number of
    bruchid species. Genes encoding for these proteins are members of the
    lectin multigene family, the most representative components being
    arcelins, phytohemagglutinins and  $\alpha$ -amylase inhibitors.
    Arcelins have been described in seven variants, some of which are
    resistance factors against the Mexican bean weevil (Zabrotes
    subfasciatus), a major bean predator. In this study the isolation and

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sequencing of arcelin genes from wild *P. vulgaris* genotypes, containing Arc3 and Arc7 variants, is reported, and similarities and evolutionary relationships among the seven known arcelins are described. The evolutionary anal. shows that arcelins 3 and 4 cluster together and are the most-ancient variants. A duplication event gave rise to two addnl. clusters, one comprising arcelins 1, 2 and 6 and separated from the cluster of arcelins 5 and 7. A multiple number of arcelin genes were found in arcelin 3 and 4 genotypes indicating that more than one type of arcelin gene may be present in the same locus. Some of these sequences are reminiscent of ancient duplication events in arcelin evolution demonstrating that arcelins have evolved through multiple duplications. A further aim of this paper was to better understand and describe the evolution of the entire lectin multigene family. Beside arcelins, a number of other types of sequences, such as putative lectins and sequences not easily classifiable, were found in genotypes containing Arc3 and Arc4. These results, together with the evolutionary anal., indicate that lectin loci are quite complex and confirm their origin by multiple duplication events.

RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 2 OF 9 CA COPYRIGHT 2004 ACS on STN

AN 139:243467 CA

TI Microarray analysis of somitogenesis reveals novel targets of different WNT signaling pathways in the somitic mesoderm

AU Buttitta, Laura; Tanaka, Tetsuya S.; Chen, Alice E.; Ko, Minoru S. H.; Fan, Chen-Ming

CS Department of Embryology, Carnegie Institution of Washington, Baltimore, MD, 21210, USA

SO Developmental Biology (San Diego, CA, United States) (2003), 258(1), 91-104

CODEN: DEBIAO; ISSN: 0012-1606

PB Elsevier

DT Journal

LA English

AB WNT signaling plays a major role in patterning the dermomyotome of the somitic mesoderm. However, knowledge of downstream target genes and their regulation is limited. To identify new genes involved in the development and early patterning of the somite, we performed a comparison of gene expression by microarray between the presomitic mesoderm and the 5 most recently formed somites of the mouse at embryonic day 9.5. We identified 207 genes upregulated and 120 genes downregulated in somite formation. Expression anal. and functional categorization of these genes demonstrate this to be a diverse pool that provides a valuable resource for studying somite development. Thus far, we have found three genes expressed in the dermomyotome of the early somite. Consistent with their expression patterns, these genes are transcriptional targets of WNT signals, but display differential **activation** by different WNTs. We further demonstrate that 1 of these genes, *Troy*, is a direct target of canonical WNT signaling, while the other 2 genes, *Selp* and *Arl4*, are not. Thus, our microarray study using microdissected tissues not only provides global information on gene expression during somite development, it also provides novel targets to study the inductive signaling pathways that direct somite patterning.

RE.CNT 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 3 OF 9 CA COPYRIGHT 2004 ACS on STN

AN 139:192518 CA

TI Gene expression profiles of human endothelial cells in response to VEGF and diagnostic and therapeutic uses thereof

IN Charnock-Jones, Stephen David; Smith, Stephen Kevin; Print, Cristin Gregor

PA Cambridge University Technical Services Ltd., UK

SO PCT Int. Appl., 84 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003066904	A2	20030814	WO 2003-GB534	20030207
	W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
	RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			

PRAI GB 2002-2881 A 20020207

AB The present invention provides methods of monitoring the progression of a disease condition associated with angiogenesis or vasculogenesis in a human subject in which a quant. determination of the transcript level of at least one gene shown in Table 1 (by which is meant one or more of any of Tables 1a to 1f) in a sample comprising cells obtained from the site of said disease is made, and compared with the transcript level of at least one gene obtained from a control sample of cells. The transcripts of Table 1 are found to respond to VEGF in a statistically significant manner under a variety of different conditions, including following serum withdrawal. The invention also provides gene chip arrays consisting of all or some of the transcripts together with appropriate controls which can be used in the methods described. In addition, the invention claims use of expression vectors for the VEGF-dependent nucleic acid sequences, use of proteins encoded the VEGF-dependent genes for drug screening, use of antibodies which bind to the proteins, and therapeutic use of antisense oligonucleotides. Quant. PCR confirmed a set of results from the Affymetrix gene array anal. SAGE (serial anal. of gene expression) identified the same subset of abundant endothelial cell transcripts as Affymetrix anal.

L11 ANSWER 4 OF 9 CA COPYRIGHT 2004 ACS on STN

AN 138:317877 CA

TI Arf, Arl, Arp and Sar proteins: A family of GTP-binding proteins with a structural device for "front-back" communication

AU Pasqualato, Sebastiano; Renault, Louis; Cherfils, Jacqueline

CS Laboratoire d'Enzymologie et Biochimie Structurales, UPR 9063 CNRS, Gif sur Yvette, 91198, Fr.

SO EMBO Reports (2002), 3(11), 1035-1041

CODEN: ERMEAX; ISSN: 1469-221X

PB Oxford University Press

DT Journal; General Review

LA English

AB A review. Arf proteins are important regulators of cellular traffic and the founding members of an expanding family of homologous proteins and genomic sequences. They depart from other small GTP-binding proteins by a unique structural device, which we call the "interswitch toggle", that implements front-back communication from the N-terminus to the nucleotide binding site. Here we define the sequence and structural determinants that propagate information across the protein and identify them in all of the Arf family proteins other than Arl6 and Arl4/Arl7. The positions of these determinants lead us to propose that Arf family members with the interswitch toggle device are **activated** by a bipartite mechanism acting on opposite sides of the protein. The presence of this communication device might provide a more useful basis for unifying Arf

homologs as a family than do the cellular functions of these proteins, which are mostly unrelated. We review available genomic sequences and functional data from this perspective, and identify a novel subfamily that we call Arl8.

RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 5 OF 9 CA COPYRIGHT 2004 ACS on STN

AN 138:135191 CA

TI Molecular diagnosis of MLL (mixed lineage leukemia), acute lymphoblastic leukemia (ALL), and acute myelogenous leukemia (AML) by gene expression profiling of related genes

IN Golub, Todd R.; Armstrong, Scott A.; Korsmeyer, Stanley J.

PA Whitehead Institute for Biomedical Research, USA; Dana-Farber Cancer Institute, Inc.

SO PCT Int. Appl., 91 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003008552	A2	20030130	WO 2002-US22823	20020717
	WO 2003008552	A3	20031211		
	WO 2003008552	C2	20040115		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2003134300 A1 20030717 US 2002-198064 20020717

PRAI US 2001-306103P P 20010717

AB The present invention relates to the diagnosis of mixed lineage leukemia (MLL), acute lymphoblastic leukemia (ALL), and acute myelogenous leukemia (AML) according to the gene expression profile of a sample from an individual, as well as to methods of therapy and screening that utilize the genes identified herein as targets. MLL is distinguished as a unique leukemia by assessing the translocations and gene expression profiling of patients diagnosed with acute lymphoblastic leukemia. It has a characteristic, highly distinct gene expression profile that is consistent with an early hematopoietic progenitor expressing select multilineage markers and individual HOX genes. Clustering algorithms reveal that lymphoblastic leukemias with MLL translocations can clearly be separated from conventional acute lymphoblastic and acute myelogenous leukemias. For the 37 samples tested, approx. 1000 genes proved underexpressed in MLL as compared to conventional ALL while approx. 200 genes were relatively highly expressed. Specifically, disclosed are 50 genes that are relatively underexpressed in MLL, and 50 genes that are relatively overexpressed in MLL (with reference GenBank provided). Thus, a distinct disease, denoted here as MLL, is proposed and the invention also shows that the differences in gene expression are robust enough to classify leukemias correctly as MLL, acute lymphoblastic leukemia or acute myelogenous leukemia. Establishing that MLL is a unique entity is critical, as it mandates the examination of selectively expressed genes for urgently needed mol. targets.

L11 ANSWER 6 OF 9 CA COPYRIGHT 2004 ACS on STN

AN 138:20443 CA
 TI Endocrine disruptor screening using DNA chips of endocrine
 disruptor-responsive genes
 IN Kondo, Akihiro; Takeda, Takeshi; Mizutani, Shigetoshi; Tsujimoto,
 Yoshimasa; Takashima, Ryokichi; Enoki, Yuki; Kato, Ikunoshin
 PA Takara Bio Inc., Japan
 SO Jpn. Kokai Tokkyo Koho, 386 pp.
 CODEN: JKXXAF
 DT Patent
 LA Japanese
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 2002355079	A2	20021210	JP 2002-69354	20020313
PRAI	JP 2001-73183	A	20010314		
	JP 2001-74993	A	20010315		
	JP 2001-102519	A	20010330		

AB A method and kit for detecting endocrine-disrupting chems. using DNA
 microarrays are claimed. The method comprises preparing a nucleic acid
 sample containing mRNAs or cDNAs originating in cells, tissues, or organisms
 which have been brought into contact with a sample containing the endocrine
 disruptor. The nucleic acid sample is hybridized with DNA microarrays
 having genes affected by the endocrine disruptor or DNA fragments
 originating in these genes have been fixed. The results obtained are then
 compared with the results obtained with the control sample to select the
 gene affected by the endocrine disruptor. Genes whose expression is
 altered by tri-Bu tin, 4-octaphenol, 4-nonylphenol, di-N-Bu phthalate,
 dichlorohexyl phthalate, octachlorostyrene, benzophenone, diethylhexyl
 phthalate, diethylstilbestrol (DES), and 17- β estradiol (E2), were
 found in mice by DNA chip anal.

L11 ANSWER 7 OF 9 CA COPYRIGHT 2004 ACS on STN

AN 137:73224 CA
 TI Method for identifying substances which positively influence inflammatory
 conditions of chronic inflammatory airway diseases
 IN Jung, Birgit; Mueller, Stefan; Kraut, Norbert
 PA Boehringer Ingelheim Pharma K.-G., Germany
 SO PCT Int. Appl., 63 pp.
 CODEN: PIXXD2

DT Patent
 LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002052270	A2	20020704	WO 2001-EP14838	20011215
	WO 2002052270	A3	20030313		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
 CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
 GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
 LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
 PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA,
 UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
 RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH,
 CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,
 BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1346228 A2 20030924 EP 2001-988031 20011215

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
 IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

US 2002150958 A1 20021017 US 2001-29654 20011221

PRAI US 2000-257878P P 20001222
 WO 2001-EP14838 W 20011215

AB The invention relates to proteins involved in inflammatory processes and
 the modulation of the function of macrophage migration

*instant
 application
 family*

inhibitory factor (MIF), defender against apoptotic cell death-1 (DAD1), ADP-ribosylation factor-like 4 (ARL4), glucosamine-6-sulphatase (GNS) and transglutaminase-2, stearyl-CoA-Desaturase, UDP glucose-ceramide glucosyltransferase in order to pos. influence inflammatory diseases.

L11 ANSWER 8 OF 9 CA COPYRIGHT 2004 ACS on STN

AN 136:226817 CA

TI Use of serum response factor (SRF) **modulator** for treating SRF related diseases

IN Nordheim, Alfred

PA Germany

SO Eur. Pat. Appl., 58 pp.

CODEN: EPXXDW

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 1186319	A1	20020313	EP 2000-119741	20000908
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
WO	2002020092	A1	20020314	WO 2001-EP10440	20010910
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU	2001087724	A5	20020322	AU 2001-87724	20010910
EP	1317310	A1	20030611	EP 2001-967325	20010910
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US	2004009913	A1	20040115	US 2003-363818	20030709
PRAI	EP 2000-119741	A	20000908		
	WO 2001-EP10440	W	20010910		
AB	The invention relates to the use of an active agent influencing the expression and/or function of SRF, SRF variants and/or members of the SRF signal transduction pathway for the preparation of a therapeutic drug or a pharmaceutical composition for the treatment of disturbances or illnesses that are linked with SRF-related cellular malfunctions. Furthermore, the invention relates to the use of a substance detecting the above signal elements for the diagnosis of disturbances or illnesses linked with SRF-related cellular malfunctions, a diagnostic kit comprising such substances and a cell line which can be used for screening and identifying active agents or substances influencing the above mentioned signal elements.				

RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 9 OF 9 CA COPYRIGHT 2004 ACS on STN

AN 129:93920 CA

TI Expression of an ADP-ribosylation factor like gene, ARF4L, is induced after transient forebrain ischemia in the gerbil

AU Katayama, Taiichi; Imaizumi, Kazunori; Tsuda, Manabu; Mori, Yasutake; Takagi, Tsutomu; Tohyama, Masaya

CS Department of Molecular Neurobiology (TANABE), Osaka University Medical School, Osaka, Japan

SO Molecular Brain Research (1998), 56(1,2), 66-75

CODEN: MBREE4; ISSN: 0169-328X

PB Elsevier Science B.V.

DT Journal

LA English

AB To elucidate the mol. mechanisms underlying post-ischemic phenomena including delayed neuronal death, the authors screened for genes which were induced in the hippocampus after transient global ischemia in the Mongolian gerbil by a differential display method, and cloned a gerbil homolog of human ADP-ribosylation factor 4L (ARF4L). Although the physiol. roles of ARF4L are unknown, it is likely that ARF4L participates in vesicle transport between the endoplasmic reticulum (ER) and Golgi complex as it contains a GTP binding site, myristoylation site and coatamer binding motif (KKXX). In situ hybridization anal. indicated that the expression of ARF4L mRNA was elevated in neurons of the dentate gyrus (DG) and CA1 regions. In DG, the signals were detected 3 h after ischemia and peaked at 6 h with subsequent gradual reduction. In the CA1 region where cell death occurs in this model, ARF4L mRNA was slightly detected from 1 to 2 days after ischemia but was absent after 3 days. Other vesicle transport-related genes such as ARF1, **ARL4** and β -COP were also induced after 5-min ischemia, suggesting that vesicle transport was **activated** in hippocampal neurons after ischemic stress. To determine the cause of the induction of ARF4L gene expression after transient ischemia, the authors examined the changes in ARF4L mRNA expression in HEK 293 cells under hypoxic conditions compared with HSP70. The expression of ARF4L mRNA was elevated at 12 h after hypoxia exposure, similarly to HSP70. These results will help to elucidate the association of upregulation of vesicle transport systems including ARF4L and stress responses of neurons after transient ischemia.

RE.CNT 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 13:47:03 ON 16 FEB 2004)

FILE 'CA' ENTERED AT 13:47:08 ON 16 FEB 2004

L1	13 S ARL4
L2	1 S ARF(W) LIKE(W) PROTEIN(W) 4
L3	2 S ARF(W) LIKE(W) 4
L4	2 S ARL(W) 4
L5	3 S ADP(W) RIBOSYLATION(W) FACTOR(W) LIKE(W) PROTEIN(W) 4
L6	5 S ADP(W) RIBOSYLATION(W) FACTOR(W) LIKE(W) 4
L7	18 S L1 OR L2 OR L3 OR L4 OR L5 OR L6
L8	151569 S MACROPHAGE OR PHAGOCY? OR MONOCY?
L9	2588082 S ACTIVAT? OR INHIBI? OR MODULAT?
L10	2660591 S L8 OR L9
L11	9 S L7 AND L10

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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

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ENTRY	SESSION
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FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 11 February 2004 (20040211/ED)

FILE RELOADED: 19 October 2003.

=> s 111

15 ARL4
3956 ARF
212 ARFS
3992 ARF
 (ARF OR ARFS)
359501 LIKE
132 LIKES
359620 LIKE
 (LIKE OR LIKES)
1363263 PROTEIN
517799 PROTEINS
1570594 PROTEIN
 (PROTEIN OR PROTEINS)
1851462 4
 0 ARF(W)LIKE(W)PROTEIN(W)4
3956 ARF
212 ARFS
3992 ARF
 (ARF OR ARFS)
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27 ARLS
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47746 ADP
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5151 RIBOSYLATION
 (RIBOSYLATION OR RIBOSYLATIONS)
741741 FACTOR
551429 FACTORS
1164710 FACTOR
 (FACTOR OR FACTORS)
359501 LIKE
132 LIKES
359620 LIKE
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 (PROTEIN OR PROTEINS)
1851462 4
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47732 ADP
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47746 ADP
 (ADP OR ADPS)

5145 RIBOSYLATION
 26 RIBOSYLATIONS
 5151 RIBOSYLATION
 (RIBOSYLATION OR RIBOSYLATIONS)
 741741 FACTOR
 551429 FACTORS
 1164710 FACTOR
 (FACTOR OR FACTORS)
 359501 LIKE
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 359620 LIKE
 (LIKE OR LIKES)
 1851462 4
 4 ADP (W) RIBOSYLATION (W) FACTOR (W) LIKE (W) 4
 106133 MACROPHAGE
 88473 MACROPHAGES
 154006 MACROPHAGE
 (MACROPHAGE OR MACROPHAGES)
 51058 PHAGOCY?
 81221 MONOCY?
 667666 ACTIVAT?
 1178120 INHIBI?
 223731 MODULAT?
 L12 3 L7 AND L10

=> d l12 1-3 bib ab

L12 ANSWER 1 OF 3 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
 AN 2003:292744 BIOSIS
 DN PREV200300292744
 TI Microarray analysis of somitogenesis reveals novel targets of different
 WNT signaling pathways in the somitic mesoderm.
 AU Buttitta, Laura; Tanaka, Tetsuya S.; Chen, Alice E.; Ko, Minoru S. H.;
 Fan, Chen-Ming [Reprint Author]
 CS Department of Embryology, Carnegie Institution of Washington, Baltimore,
 MD, 21210, USA
 fan@ciwemb.edu
 SO Developmental Biology, (June 1 2003) Vol. 258, No. 1, pp. 91-104. print.
 ISSN: 0012-1606 (ISSN print).
 DT Article
 LA English
 ED Entered STN: 25 Jun 2003
 Last Updated on STN: 25 Jun 2003
 AB WNT signaling plays a major role in patterning the dermomyotome of the
 somitic mesoderm. However, knowledge of downstream target genes and their
 regulation is limited. To identify new genes involved in the development
 and early patterning of the somite, we performed a comparison of gene
 expression by microarray between the presomitic mesoderm and the 5 most
 recently formed somites of the mouse at embryonic day 9.5. We identified
 207 genes upregulated and 120 genes downregulated in somite formation.
 Expression analysis and functional categorization of these genes
 demonstrate this to be a diverse pool that provides a valuable resource
 for studying somite development. Thus far, we have found three genes
 expressed in the dermomyotome of the early somite. Consistent with their
 expression patterns, these genes are transcriptional targets of WNT
 signals, but display differential **activation** by different WNTs.
 We further demonstrate that 1 of these genes, *Troy*, is a direct target of
 canonical WNT signaling, while the other 2 genes, *Selp* and *Arl4*,
 are not. Thus, our microarray study using microdissected tissues not only
 provides global information on gene expression during somite development,
 it also provides novel targets to study the inductive signaling pathways
 that direct somite patterning.

L12 ANSWER 2 OF 3 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
 AN 2003:67411 BIOSIS
 DN PREV200300067411
 TI Arf, Arl, Arp and Sar proteins: A family of GTP-binding proteins with a structural device for 'front-back' communication.
 AU Pasqualato, Sebastiano; Renault, Louis; Cherfils, Jacqueline [Reprint Author]
 CS Laboratoire d'Enzymologie et Biochimie Structurales, UPR 9063 CNRS, 1 Avenue de la Terrasse, 91198, Gif sur Yvette Cedex, France
 cherfils@lebs.cnrs-gif.fr
 SO EMBO Reports, (November 2002) Vol. 3, No. 11, pp. 1035-1041. print. ISSN: 1469-221X (ISSN print).
 DT Article
 LA English
 ED Entered STN: 29 Jan 2003
 Last Updated on STN: 29 Jan 2003
 AB Arf proteins are important regulators of cellular traffic and the founding members of an expanding family of homologous proteins and genomic sequences. They depart from other small GTP-binding proteins by a unique structural device, which we call the 'interswitch toggle', that implements front-back communication from the N-terminus to the nucleotide binding site. Here we define the sequence and structural determinants that propagate information across the protein and identify them in all of the Arf family proteins other than Arl6 and **Arl4**/Arl7. The positions of these determinants lead us to propose that Arf family members with the interswitch toggle device are **activated** by a bipartite mechanism acting on opposite sides of the protein. The presence of this communication device might provide a more useful basis for unifying Arf homologs as a family than do the cellular functions of these proteins, which are mostly unrelated. We review available genomic sequences and functional data from this perspective, and identify a novel subfamily that we call Arl8.

L12 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
 AN 1998:350438 BIOSIS
 DN PREV199800350438
 TI Expression of an ADP-ribosylation factor like gene, ARF4L, is induced after transient forebrain ischemia in the gerbil.
 AU Katayama, Taiichi [Reprint author]; Imaizumi, Kazunori; Tsuda, Manabu; Mori, Yasutake; Takagi, Tsutomu; Tohyama, Masaya
 CS Dep. Mol. Neurobiol., Osaka Univ. Med. Sch., 2-2 Yamadaoka, Suita, Osaka 565, Japan
 SO Molecular Brain Research, (May, 1998) Vol. 56, No. 1-2, pp. 66-75. print. CODEN: MBREE4. ISSN: 0169-328X.
 DT Article
 LA English
 ED Entered STN: 13 Aug 1998
 Last Updated on STN: 10 Sep 1998
 AB To elucidate the molecular mechanisms underlying post-ischemic phenomena including delayed neuronal death, we screened for genes which were induced in the hippocampus after transient global ischemia in the Mongolian gerbil by a differential display method, and cloned a gerbil homologue of human ADP-ribosylation factor 4L (ARF4L). Although the physiological roles of ARF4L are unknown, it is likely that ARF4L participates in vesicle transport between the endoplasmic reticulum (ER) and Golgi complex as it contains a GTP binding site, myristoylation site and coatmer binding motif (KKXX). In situ hybridization analysis indicated that the expression of ARF4L mRNA was elevated in neurons of the dentate gyrus (DG) and CA1 regions. In DG, the signals were detected 3 h after ischemia and peaked at 6 h with subsequent gradual reduction. On the other hand, in the CA1 region where cell death occurs in this model, ARF4L mRNA was slightly detected from 1 to 2 days after ischemia but was absent after 3 days. Other vesicle transport-related genes such as ARF1, **ARL4** and

beta-COP were also induced after 5-min ischemia, suggesting that vesicle transport was **activated** in hippocampal neurons after ischemic stress. To determine the cause of the induction of ARF4L gene expression after transient ischemia, we examined the changes in ARF4L mRNA expression in HEK 293 cells under hypoxic conditions compared with HSP70. The expression of ARF4L mRNA was elevated at 12 h after hypoxia exposure, similarly to HSP70. These results will help to elucidate the association of upregulation of vesicle transport systems including ARF4L and stress responses of neurons after transient ischemia.

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TOTAL

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